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**Issue Date: 15 April 2003**

CASE NO.: **2001-LHC-2976**

OWCP NO.: **06-184461**

In the matter of:

**WILLIAM SAULS**
Claimant,
v.
**JACKSONVILLE SHIPYARDS, INC.**
and
**CNA CONTINENTAL CASUALTY COMMERCIAL INSURANCE**,
and
**ST PAUL INSURANCE COMPANY, INC.**,
and
**TRAVELERS CASUALTY & SURETY**,
Employer/Carriers,
and
**GIBBS SHIPYARD/RAWLS BROTHERS CONTRACTORS**,
and
**OHIO CASUALTY**,
Employers/Carrier,
and
**DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,**
Party-In-Interest.

Representation:

Melissa Olson, Esq., for the claimant
Bonnie Murdoch, Esq., for ACE/INA and ARM
Robert Sharp, Esq., for AEROJET and REM
Carolyn Blue, Esq., for ARM and St. Paul Fire and Marine
Dale Stone, Esq., for CNA Commercial Insurance
Clayton Harland, Esq., for Travelers Insurance
Phillip Giannikas, Esq., for the Director, OWCP

## DECISION AND ORDER DENYING BENEFITS

This proceeding arises from a claim filed under the provisions of the Longshore and Harbor Workers Compensation Act, as amended, 33 U.S.C. 901 et seq.

A formal hearing was held in Jacksonville, Florida on May 14, 2002 at which time all parties were afforded full opportunity to present evidence and argument as provided in the Act and the applicable regulations.

The findings and conclusions which follow are based upon a complete review of the entire record in light of the arguments of the parties, applicable statutory provisions, regulations and pertinent precedent.

## Preliminary Matters[1]

At the hearing, counsel for ACE/INA and counsel for Aerojet and REM raised motions for summary decisions on behalf of their parties. There were no objections. Therefore, ACE/INA, Aerojet, and REM were dismissed as parties in the case. (TR. 9-12).

Motions for summary decision on behalf of St. Paul Fire and Marine and on behalf of CNA received objections from numerous parties. Therefore, the motions were denied at that time. (TR 12-24).

There was extensive post hearing development with depositions and with tissue block studies.

At the hearing, the claimant submitted exhibits CX 1-13, the Director DX 1-7, St. Paul's SX 1-5, CNA submitted CNAX 1, and Travelers submitted TX 1-6 and 9 and 12. These exhibits were entered into the record.

Post hearing the parties submitted

JX 1 An article titled "Report of the Pneumoconiosis Committee"

The Director submitted

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[1] The following abbreviations will be used as citations to the record:

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|---|---|---|
| JS | - | Joint Stipulations; |
| TR | - | Transcript of the Hearing; |
| CX | - | Claimant's Exhibits; |
| DX | - | Director's Exhibits; |
| SX | - | St. Paul's Exhibits; |
| CNAX | - | CNA's Exhibits; |
| TX | - | Travelers' Exhibits; and |
| JX | - | Joint Exhibit. |

DX 8 July 2002 deposition of Robert N. Jones, M.D.

DX 9 August 2002 deposition of Philip Cagle, M.D.

DX 10 Digestion study by Dr. Cagle

The claimant submitted

CX 14 March 2002 report from Eugene Mark, M.D.

CX 15 CV of Dr. Mark

CX 16 July 2002 deposition of Douglas Pohl, M.D.

CX 17 June 2002 deposition of Arthur DeGraff, M.D.

These additional exhibits are entered into the record.

## Stipulations

The Claimant and the Employers/Carriers have stipulated to the following:

1. That the parties are subject to the jurisdiction of the Longshore and Harbor Workers' Compensation Act;

2. An Employer/Employee relationship existed at all relevant times;

3. The claimant worked for Jacksonville Shipyard from 1969 until he was laid off on August 25, 2989;

4. Sauls took voluntary retirement on October 25, 1989;

5. The claimant is married and has a handicapped child. (JS) (TR. 52).

## Issues

1. Whether the claimant has work related asbestosis?

2. Whether the claimant gave timely notice of the impairment?

3. Timeliness of notice of medical treatment?

4. Which party is the responsible Employer/Carrier?

5. Entitlement to Section 8(f) relief?

## Presence or absence of asbestosis

Claimant's counsel states that Mr. Sauls was exposed to asbestos and welding fumes for some thirty years while working in the shipyard.

Mr. Sauls was exposed to asbestos almost everyday, since most of his rigging work involved the rip out and removal of asbestos.

Mr. Sauls was promoted to a yard superintendent in the mid-1970's when Freuhauf took over Jacksonville Shipyards. After the mid 1970's, Mr. Sauls was involved in upgrading the yard structures and buildings, and was no longer exposed to asbestos.

The claimant states that the employer offered no evidence refuting or contradicting Mr. Saul's extensive exposure to asbestos and other lung irritants. Indeed biopsy of his lungs found asbestos still embedded in his lungs thirty years later, thus offering objective proof of his exposure.

In July of 1998, Mr. Sauls was found to have lung cancer. Mr. Sauls' lung capacity and pulmonary function were drastically restricted. (CX 4) Chest x-rays taken at Baptist Medical Center revealed significant bilateral pleural thickening and interstitial fibrosis. He was forced to endure a left lung lobectomy and radiation therapy. In 2000, Mr. Saul developed a recurrent cancer. Dr. James Krainson reviewed Mr. Sauls chest x-rays and confirmed the interstitial lung disease, or asbestosis and pleural thickening and pleural plaques, which were previously noted on the Baptist Medical Center x-rays. (CX 8).

Claimant's counsel notes that there are numerous medical opinions in this case. It is argued that

> The better and more logical line of reasoning is asserted by Drs. Mark, DeGraff, Pohl and Hammar, that asbestosis and asbestos-related lung cancer are separate and distinct diseases. While both are caused by asbestos they are not related. Sun exposure can cause skin cancer and tanning, but not all persons suffering from skin cancer are tan. A diagnosis of asbestosis is not necessary for the development of asbestos-related lung cancer.

The Employer/Carriers and the Director state, in essence, that

> Drs. Jones, Cagle, Mark and Hammar possess a degree of specialized academic and research knowledge and experience that sets them apart from the other physicians whose opinions and reports are in the record. Accordingly, with respect to the general subject of the relationship between asbestos exposure and lung cancer, these physicians' opinions should be recognized as more authoritative than the opinions of the other physicians.
>
> Out of these four expert physicians whose ongoing and specialized fields of research involve occupational diseases of the lung, Drs. Jones and Cagle subscribe to the asbestosis-as-a-reliable-marker approach to diagnosing a causal connection between asbestos exposure and lung cancer, Dr. Mark subscribes to the contrary view, and Dr. Hammar's views on the subject are not readily apparent.
>
> There exists, in current medical science, two schools of thought exist with regard to the minimal levels of asbestos exposure necessary to cause, aggravate, or contribute to lung cancer. These two schools are reflected in the conflicting opinions of the "defendants" and the claimant's medical experts.
>
> Drs. Cagle and Jones expressed reasoned medical opinions that a diagnosis of asbestosis must be present in order to establish a causal connection between asbestos exposure and lung cancer (DX 3, pp. 5-10; DX 5, pp. 5-7). The opinions do not recognize that all exposures to asbestos, regardless of their magnitude, are carcinogenic. Rather, before asbestos can be found to have led to lung cancer in an exposed individual, the person exposed must have incurred an exposure of a sufficient minimal magnitude (DX 9, pp. 9-10). This exposure of sufficient magnitude translates into a minimal "threshold" level of asbestos exposure (DX 3, p. 8; DX 9, pp. 9—10).

The "defendants" stated that

> The opposing school of thought represented to a greater or lesser degree by the claimant's experts considers the presence of the disease of asbestosis as

> unnecessary to establish a causal connection between asbestos exposure and lung cancer. This opposing school of thought accepts a mere history of asbestos exposure—usually, as that history is verbally related to a medical provider by the person supposedly exposed and without any corresponding objective verification or quantification of its magnitude—as being the most reliable indicator of a causal relationship between asbestos exposure and cancer. Implicit in this school of thought is the notion that there is no minimal threshold level of asbestos exposure necessary to lead to lung cancer.

## Evaluation of the Evidence

Clinical data at Baptist Hospital in July 1998 indicated that while Sauls was being worked up for knee surgery, a chest X-ray revealed a mass in the left lung. It was reported that he had been exposed to asbestos for many years, and that he smoked two packs of cigarettes a day for forty years.

Dr. Trent reported that studies revealed bronchogenic carcinoma. The claimant underwent a resection of the left lung. In mid-2000, it was reported that there had been a recurrence and that Sauls was being treated with chemotherapy and radiation therapy. (CX 4).

Dr. Johnson, a radiation oncologist, provided treatment from September 1998 through late 2000. (CX 5). A May 2002 report is in the record. (CX 13).

The claimant was deposed in December 2000. He testified that he began working as a rigger for Gibbs Shipyard in 1953. He worked around insulation containing asbestos. The shipyard changed names frequently and he worked in the city as well as at Mayport.

The lung problem was noted during the pre-surgical work up for the knee. A physician advised him that the lung cancer was due to asbestos. (CX 11, p. 34).

On January 23, 2001, Sauls filed a claim for benefits based on exposure to asbestos resulting in lung cancer. (TX 3).

In July 2000, Dr. Krainson, a B-reader, reported a November 1998 chest X-ray as 1/0 for small opacities.

The X-ray revealed

Interstitial Lung Disease Consistent with Asbestos Exposure / Asbestos Related Disease Pleural Thickening / Pleural Plaques consistent with Asbestos Exposure / Asbestos Related Disease. (CX 8).

In March 2001, Dr. Mark, a pathologist, reported that he had reviewed slides from the lung resection in September 1998. The physician stated that

> Prussian blue stain shows asbestos bodies on each of the four slides that contain nontumorous lung. The other two slides contain only tumor and lymph node. The asbestos bodies are long and blue and beaded and have thin translucent cores. There is approximately one asbestos body per square centimeter of non-tumorous lung.

Dr. Mark reported that

> The patient smoked cigarettes for many years and then quit smoking approximately twenty-three years prior to detection of his carcinoma of the lung according to a letter from Brown, Terrell, Hogan, Ellis, McClamma & Yegelwel to me dated 22 January 2001. The patient was exposed to asbestos according to the medical records and according to the same letter from Brown, Terrell, Hogan, Ellis, McClamma & Yegelwel to me.
>
> Cigarette smoke and asbestos each can cause carcinoma of the lung including squamous cell carcinoma. Together, cigarette smoke and asbestos act in a synergistic manner to cause carcinoma of the lung. All of the types of asbestos can cause carcinoma of the lung. All of the exposures to asbestos which occur prior to the development of the carcinoma contribute to its pathogenesis.
>
> I conclude that the patient has developed a squamous cell carcinoma of the lung. I conclude that he has inhaled asbestos. I conclude that the cigarettes which he reportedly smoked and the asbestos which he inhaled together caused the squamous cell carcinoma of the lung. I conclude that the contribution of the cigarettes would be greater if he had continued to smoke up until the time of development of the squamous cell carcinoma than if he had stopped smoking many years previously. I conclude that the

squamous cell carcinoma has recurred in the chest wall. (CX 14).

In August 2001, Dr. Mark reported that he had reviewed slides from a May 2001 vocal cord biopsy. The physician indicated that

> Histopathologically, the squamous cell carcinoma in the larynx differs from the previously resected squamous cell carcinoma in the lung. The former is well differentiated and superficia1 on a muscosal surface. The latter is poorly differentiated and deeply invasive within the lung. The clinical and radiographic features together with the pathologic findings indicate that the squamous cell carcinoma of the lung and the squamous cell carcinoma of the larynx are two independent primary malignancies.
>
> Smoking and asbestos each can cause or contribute to cause carcinoma of the larynx including squamous cell carcinoma.
>
> I conclude that the patient has developed two squamous cell carcinomas, first one in the lung and then one in the larynx. I conclude that the cigarettes which he reportedly smoked and the asbestos which he inhaled caused both squamous cell carcinomas. (CX 3).

Dr. Pohl, who is board certified in pathology, reviewed records and slides in August 2001. The physician stated, in part

> Mr. Sauls′ asbestos exposure was prolonged and significant, fulfilling the Helsinki Consensus Conference criteria for significant occupational exposure to asbestos. Mr. Sauls′ x-rays and pathology slides show objective evidence of his past asbestos exposure in the form of dense interstitial fibrosis diagnostic for pulmonary asbestosis. Pulmonary asbestosis arises as a result of the irritant effect of inhaled asbestos fibers on the lung parenchyma. Asbestosis is a disease that is typically seen of only in individuals with past heavy asbestos exposure. As a result, Mr. Sauls′ asbestosis confirms his past asbestos exposure and indicates that his exposure to asbestos was in fact quite heavy.
>
> Since asbestos related lung cancer is a dose dependent disease, with higher lung cancer risk

> occurring with more significant asbestos exposure, it is clear that Mr. Sauls' asbestos exposure greatly increased his lung cancer risk. Given these facts, it is my opinion, within a reasonable degree of medical certainty, that Mr. Sauls' past occupational exposure to asbestos was the principal cause of his lung cancer. (CX 2).

Dr. DeGraff, who is board certified in internal medicine and in pulmonary disease, reviewed records in September 2001 and noted the "B"-reading of the November 1998 X-ray. It was also noted that

> Dr. Pohl indicates that Mr. Sauls had been a smoker in past years but had stopped 20 years prior to diagnosis of his lung cancer. He also indicated that there was a significant past heavy asbestos exposure and that chest x-rays revealed "objective evidence of past asbestos exposure in the form of both pleural and parenchymal fibrosis." He further indicates that asbestos-related lung cancer is a dust-dependent disease with the incidence of lung cancer occurring proportionate to asbestos exposure. He further indicates that since Mr. Sauls had stopped smoking 20 years before his cancer was diagnosed, his abstinence from smoking should significantly reduce the lung cancer risk associated with smoking. Indeed studies have shown that by 20 years after smoking cessation, the risk of lung cancer secondary to smoking is approaching the risk of that lifelong non-smokers have of developing lung cancer. Therefore I agree with Dr. Pohl that Mr. Sauls' occupational exposure to asbestos was clearly the primary cause of development of his lung cancer.
>
> Dr. Pohl then goes on to review the pathologic slides which are described by the pathology department in Mr. Sauls' records. In addition he indicates that in slide case S-98-10234, which is of lung tissue removed in left thoracotomy on 9/28/98, there is evidence of well-advanced interstitial fibrosis consistent with a diagnosis of asbestosis.
>
> Mr. Sauls' records were also reviewed by Dr. Eugene Mark of Massachusetts General Hospital. Dr. Mark is a pathologist. He describes the tissue block of tumor and indicates that he re-cut sections from the six blocks of tissue that were imbedded in paraffin and

indicates that there is “desmoplastic fibrosis around the tumor and also shows old interstitial fibrosis of alveolar walls and some regions distant from the tumor.” He further indicates that “Prussian blue stain shows asbestos bodies on each of the four slides that contain non-tumerous lung.” Dr. Mark then goes on to conclude that Mr. Sauls had a squamous cell carcinoma of the lung and that he had inhaled asbestos. He also concludes that cigarette smoking and asbestos act in a synergistic manner to cause carcinoma of the lung.

Dr. DeGraff then stated

> As you know, it has been suggested that the synergism between cigarette smoking and asbestos is most likely when the exposure to cigarette smoke and asbestos occur contemporaneously. It has been hypothesized that this is in part because of the absorbent nature of asbestos which causes carcinogens from the cigarette smoke to be absorbed and concentrated on the asbestos fibers which then migrate into lung tissue.
>
> In conclusion, it is my opinion that Mr. Sauls has asbestosis which has been documented both radiologically and pathologically. While development of asbestosis does not necessarily occur in persons with heavy asbestos exposure, its presence confirms the history of heavy asbestos exposure. It is further my opinion that Mr. Sauls′ lung cancer developed as a consequence of past heavy asbestos exposure. In view of his recently diagnosed recurrence of lung cancer, and his recent radiation therapy begun on 8/8/00 and completed on 9/5/00, Mr. Sauls is 100% disabled according to the AMA Guide to Evaluation of Permanent Impairment, 5th Edition. The 100% disability rating should continue until September 5, 2001 if there is no evidence of recurrent lung cancer by that time. Otherwise the 100% disability rating should continue from the time of new recurrence of lung cancer until Mr. Sauls′ death. (CX 1).

In April 2002, Dr. Cagle, who is board certified in pathology, reviewed slides and reports.

**Microscopic**:

Slides from the left upper lobectomy labeled S98-10234 show lung in which there is a non-small cell carcinoma predominantly with features of a poorly differentiated to moderately differentiated squamous cell carcinoma with focal suggestion of gland formation. Lung parenchyma uninvolved by tumor shows bullous emphysema with destruction of alveolar parenchyma leaving residual islands of fibrovascular tissue, focal remodeled tissue and bullae. A few microscopic foci of peribronchiolar fibrosis consistent with changes from previous tobacco smoking are present. No asbestos bodies are present on H&E or iron stained sections. The remaining slides consist of various small biopsies and cytology specimens: NG-98-00745 shows cells of nonsmall cell carcinoma, S01-004812 shows a keratinizing squamous cell carcinoma, S98-07802 shows bronchial mucosa and wall and squamous cell carcinoma, NG-98-00746 shows cells of nonsmall cell carcinoma, S-00-006717 shows bronchial mucosa and wall and FD-00-00189 shows a squamous cell carcinoma. No lung parenchyma is present in any of these slides. No asbestos bodies are present in any of these slides.

The physician stated, in part

Mr. Sauls had emphysema in his lung tissue due to tobacco smoke exposure. This emphysema can be seen by direct examination of samples of Mr. Sauls′ actual lung tissue under the light microscope. Within reasonable medical probability, Mr. Sauls′ emphysema puts him in the category of smokers most likely to get lung cancer from exposure to tobacco smoke.

...(6) Within reasonable medical probability, the fibrous tissue seen in Mr. Sauls′ lungs was not caused, contributed to or aggravated by exposure to asbestos.

In his report of 8-30-01. Dr Pohl makes mention of interstitial fibrosis in the slides with Mr. Sauls′ lung tissue from the lobectomy (S98-10234) that he interprets as “consistent with pulmonary asbestosis.” Dr. Pohl does not mention finding asbestos bodies and, indeed, no asbestos bodies are present in the H&E stained slides of Mr. Sauls′ lung tissue from the lobectomy. H&E is the routine stain for tissue and asbestos bodies can be seen on H&E sections. However, iron stain makes asbestos bodies easier to see and is

used to enhance searching for asbestos bodies in lung tissue sections. An iron stain was performed on Mr. Sauls' lung tissue sample at Baylor College of Medicine and no asbestos bodies are present in Mr. Sauls' lung tissue on iron stain. Dr. Pohl did not perform an iron stain on Mr. Sauls' lung tissue.

Asbestosis is fibrosis or scarring of the interstitium (lining of the alveoli or air sacks) in the lung parenchyma (meat of the lung where the alveoli are located and gas exchange occurs). Asbestosis is caused by very high levels of exposure to asbestos. The minimal levels of asbestos exposure at which asbestosis may develop are 1000 asbestos bodies per gram of wet weight lung tissue on a digestion study or 25 fibers per cc-year in industrial hygiene terms. Most actual patients with asbestosis have much higher levels of exposure than these minimal amounts. The presence of asbestos bodies is necessary for the diagnosis of fibrosis due to asbestos exposure (See Churg, A: Update on Asbestos Pahtology 2002, International Update on Occupational and Environmental Respiratory Disease, Houston, TX, March 8-10, 2002; Roggli VL, Oury T. Interstitial fibrosis, predominantly mature. In: Cagle PT (ed) Diagnostic pulmonary pathology. New York: Marcel Dekker, 2000: 77-101; Pathology of Occupational Lung Disease, Churg A, Green FHY, editors, 2nd edition, Baltimore: Williams & Wilkins, 1998; Roggli VL, Greenberg SD, Pratt PC (eds) Pathology of Asbestos-Associated Diseases. Boston: Little, Brown & Company, 1992).

Within reasonable medical probability, in the absence of asbestos bodies, the fibrous tissue seen in Mr. Sauls' lungs was not caused, contributed to or aggravated by exposure to asbestos.

... Examination of Mr. Sauls' actual lung tissue under the light microscope does show some minimal fibrosis which is anatomically related to his bullous emphysema and areas affected by tobacco smoke and has the histopathologic appearance of smoking-related changes. Within reasonable medical probability, the fibrous tissue seen in Mr. Sauls' lungs was caused by exposure to tobacco smoke.

... In Mr. Sauls' case, as already discussed, no asbestos bodies are present by direct examination of his actual lung tissue under the light microscope. Even if he had some amount of asbestos exposure on his job, Mr. Sauls did not have enough exposure for it to show up as asbestos bodies in his lung tissue slides. If Mr. Sauls did not have enough asbestos exposure for it to show up as asbestos bodies in his lung tissue slides, then Mr. Sauls did not have enough asbestos exposure to cause lung cancer. Within reasonable medical probability, in the absence of asbestos bodies, Mr. Sauls' lung cancer was not caused, contributed to or aggravated by exposure to asbestos.

In Summary:

1. Within reasonable medical probability, Mr. Sauls' lung cancer was caused by exposure to tobacco smoke.

2. Within reasonable medical probability, the presence of emphysema in Mr. Sauls' lung tissue puts him in the category of smokers most likely to get lung cancer from exposure to tobacco smoke and provides further proof that tobacco smoking caused Mr. Sauls' lung cancer.

3. Within reasonable medical probability, the fact that Mr. Sauls developed another tobacco-related cancer, cancer of the larynx, is further evidence that Mr. Sauls' lung cancer was caused by tobacco smoke.

4. Within reasonable medical probability, Mr. Sauls was still at risk for lung cancer from exposure to tobacco smoke even 18 years after he quit smoking.

5. Within reasonable medical probability, exposure to tobacco smoke was sufficient by itself to cause Mr. Sauls' lung cancer without additional contribution from asbestos or any other agent.

6. Within reasonable medical probability, the fibrous tissue seen in Mr. Sauls' lungs was not caused, contributed to or aggravated by exposure to asbestos.

7. Within reasonable medical probability, the fibrous tissue seen in Mr. Sauls' lungs was caused by exposure to tobacco smoke

8. Within reasonable medical probability, in the absence of asbestosis, Mr. Sauls' lung cancer was not caused, contributed to or aggravated by exposure to asbestos.

9. Within reasonable medical probability, in the absence of asbestos bodies, Mr. Sauls' lung cancer was not caused, contributed to or aggravated by exposure to asbestos.

Diagnosis:

Lung, left upper lobe, lobectomy

—-Non-small cell carcinoma

--Bullous emphysema with tobacco related focal peribronchiolar fibrosis

(DX 3; TX 12).

Dr. Jones, who is board certified in internal medicine and in pulmonary disease, reviewed records in May 2002. The physician noted that Dr. Mark had reviewed materials but that review of the lung sections was not provided to Dr. Jones. Drs. Pohl and DeGraff had referred to the lung section review by Dr. Mark.

Dr. Jones reported that Dr. Pohl

> stated that lung tissue distant from the cancer shows "advanced well-established interstitial fibrosis consistent with pulmonary asbestosis." However, he did not describe asbestos bodies, without which there is no valid histopathological diagnosis of asbestosis. ... Dr. DeGraff concluded that asbestosis proved heavy exposure in Mr. Sauls, and that his cancer developed as a consequence.

Dr. Jones reviewed several X-rays and CT scans. The physician reported

> In this case (assuming the accuracy of the second-hand report of Dr. Mark's findings) consulting pathologists differ on the question of whether Mr. Sauls' lung tissue meets histopathologic criteria for asbestosis. My only contributions in that dispute are the following observations.
>
> 1) The imaging studies show no sign of asbestosis, and they don't even show a pleural plaque. Plaques develop after much lower exposures than are required to cause asbestosis, so plaques are evident (on CT scans) in the great majority of cases of asbestosis.
>
> 2) Between the two consulting pathologists who adhered to accepted criteria for diagnosis (or rejection) of asbestosis, Dr. Cagle has an international reputation as an authority on the pathology of asbestos-related diseases. His report also clearly states the requirement for a diagnosis of asbestosis, as opposed to a mere history of exposure, to attribute lung cancer to asbestos.
>
> Concerning whether Mr. Sauls was at low risk of lung cancer from his past cigarette use, the Surgeon General's reports indicate that elevated risk persists for long periods after smoking cessation. Sir Richard Doll, the world's foremost authority on smoking and lung cancer, believes that the risk elevation persists for life.
>
> (DX 5, TX 9).

When deposed in June 2002, Dr. DeGraff testified that he specialized in pulmonary medicine. The physician placed emphasis on Dr. Mark's pathologic examination, and the indication that Dr. Cagle did not examine the slides used by Dr. Mark.

Dr. DeGraff agreed that a diagnosis of asbestosis was not necessary to relate asbestos exposure to the development of lung cancer. The physician noted that Dr. Mark had described the presence of ferruginous bodies. Dr. DeGraff acknowledged that he did not review the X-rays.

Dr. DeGraff stated

> The issue is a definitive diagnosis as opposed to more probable than not, and I think we're dealing with

more probable than not here. But also, here we have pulmonary fibrosis and we have asbestos bodies and we have a history of exposure. So you have three things which would lead you to the diagnosis of asbestosis. And in any case, my opinion is that asbestosis is a separate issue. The patient has lung cancer. He has a heavy asbestos exposure. The asbestosis only indicates the degree that he had heavy asbestos exposure. The lung cancer develops independent of the pulmonary fibrosis. It is not a scar cancer. It develops within the airways. (CX 17, pp. 60 and 61).

Later, the physician reported

pulmonary fibrosis due to asbestos is asbestosis. We're all saying that the pulmonary fibrosis is due to asbestos here. And that's my definition of asbestosis. If you want to take another definition of asbestosis, that's fine with me. But the issue is that he had pulmonary fibrosis, and that's a marker for high levels of asbestos exposure. And we're all talking about a high level of -- the level of asbestos exposure. And the presence or absence of whether it's asbestosis or not is immaterial. We're looking for markers of high levels of asbestos exposure, and I think we have markers of high levels of asbestos exposure in the pulmonary fibrosis, in the ferruginous bodies, and in history. (CX 17, p. 65).

When deposed in July 2002, Dr. Pohl testified that

The principal non-malignant disease is asbestosis. It's a fibrosing disease of the lung caused by the inhalation of asbestos dust.

... The corollary of lung fibrosis is pleural fibrosis, and these patients can also develop pleural plaques when the asbestos fibers leave the lungs and go out in the pleura of the chest cavity. (CX 16, p.8).

Asbestos is the mineral fiber that causes asbestosis, which is the fibrosing process in the lung. The definition of asbestosis is the presence of an interstitial fibrosis seen under the microscope, usually in the presence of asbestos fibers or bodies.

... Virtually all of (the asbestos dust) that's inhaled is exhaled, normally. So only a small

proportion of it is retained in the lungs. And even the small proportion that's retained in the lungs is eventually removed through the body's normal defense mechanisms. (CX 16, p.9).

Dr. Pohl testified that the slides showed lung cancer as well as the presence of an interstitial fibrosis.

The pattern of this fibrosis is quite typical of pulmonary asbestosis; and by that I mean it's a diffuse fibrosis, it extends in a peribronchial location, between respiratory units. By definition that's what we look for when we make the diagnosis of asbestosis. (CX 16, p. 25).

Cigarette smoking fibrosis was seen in areas of emphysema, but

that type of fibrosis is patchy and irregular. The fibrosis of asbestosis is more confluent. (P.25).

Dr. Pohl was asked

Q. If you had not had a chance to review his pathological materials and you didn't know whether there were any asbestos bodies in there or not, would you have an opinion as to whether or not that history that I gave you hypothetically (of some 20 years exposure) would have been a significant enough exposure to asbestos to contribute to cancer of the human lung?

A. Yes. The history in and of itself was enough to attribute his lung cancer to that exposure. (p.35).

Q. Did you need to find the asbestos body in the lung in order to make a determination as to whether or not his past exposure to asbestos contributed to his lung cancer?

A. No.

Q. And why is that?

A. Because again it's recognized that asbestos fibers disappear with time. So if you look for them many years after an individual ceased working around asbestos you won't find them. The Helsinki Consensus

> Conference, which met in Finland several years ago, acknowledged the fact that the occupational history is the best evidence of an individual's past exposure to asbestos. (CX 16, pp. 35 & 36).

Dr. Pohl reported that Dr. Cagle has a

> double standard where he requires asbestos bodies to be demonstrated to attribute a lung cancer to asbestos exposure, but he doesn't require emphysema to be present to attribute somebody's lung cancer to smoking. And in fact Dr. Cagle freely accepts a smoking history as sufficient evidence, but he will not accept a history of asbestos exposure as sufficient evidence. (p. 43).

When asked about Dr. Mark's reluctance to diagnose asbestosis in view of a small fiber count, Dr. Pohl stated

> That would surprise me because that runs contrary to all the published literature on this subject. (p. 71).

Dr. Pohl relied on the Helsinki Consensus Conference Report which indicated

> that the most reliable source of information concerning an individual's exposure is his occupational history. They go on to say that x—ray findings tend to be inaccurate, and that pathology is not always available or sufficient to make the determination of prior exposure. (pp. 75 & 76).

Dr. Pohl indicated that records and the claimant's depositions indicated that he was last exposed to asbestos about 1975 or 1976. (p. 86).

The physician was asked

> Q. Doctor, in your consultative opinion in this case you did not discuss a specific pattern of fibrosis which led you to believe that asbestos was involved as opposed to other patterns of fibrosis, did you?
>
> A. That's incorrect. Quoting from Page 3 I stated that lung tissues sampled distant from the tumor shows the

presence of an advanced, well-established interstitial fibrosis consistent with pulmonary asbestosis.

Q. In that statement, however, there's no speaking of a certain pattern; you refer to some kind of pattern which was unique to asbestos. Correct?

A. Well, I didn't go on to describe it in great detail paragraph after paragraph, but the fact that I said that the appearance of it was consistent with asbestosis indicates that I believe that pattern was present. (CX 16, pp. 87 & 88).

Dr. Pohl stated that his report indicated that

lung tissues sampled distant from the tumor shows the presence of an advanced, well-established interstitial fibrosis consistent with pulmonary asbestosis. (pp. 88 & 89).

Dr. Pohl was asked

Q. And if I understand correctly, your diagnosis of asbestosis is based upon the particular pattern of fibrosis that you see as unique to asbestosis—related fibrosis.

A. In Mr. Sauls' case, his pathology actually meets the CAP-NIOSH 1982 criteria for the diagnosis of asbestosis: the presence of an interstitial fibrosis extending between bronchiolar respiratory units and the observation of at least two asbestos bodies that is present in those tissues.

Q. Sir, the fact is, though, in your consultative report you did not state that, did you?

A. I did not have available paraffin blocks to do iron stains. That information has now become available to me since I issued my report, and my opinion now, as of today, is that this man meets the criteria, the CAP-NIOSH criteria, for pulmonary asbestosis. (p. 94).

It was pointed out that Dr. Craighead, in the CAP-NIOSH report, indicated that two fibers had to be present in a sample for a diagnosis of asbestosis. Reference was made to Dr. Churg's article which stated that

> A report prepared by the Pneumoconiosis Committee of the American College of Pathologists proposed that at least two asbestos bodies needed to be observed before one could make a diagnosis of asbestosis. This precaution was imposed to avoid finding a single body from background atmospheric exposure and thus labeling another cause of interstitial fibrosis as asbestosis. (CX 16, p.99).

Dr. DeGraff was deposed in late June 2002 and testified that he had reviewed depositions and medical records. The physician stated that asbestos particles could lead to pleural plaques and to pulmonary fibrosis. Dr. DeGraff reported that

> Asbestosis is somewhat unusual in that the —— despite the possibility of scar tissue occurring within the lung parenchyma, the asbestos—related lung cancers tend to occur within the airways, within the conducting airways ... (which) are located throughout the lung tissue, and this is why the -- why many of us feel that scarring —— lung parenchymal scarring or asbestosis is not necessary for the development of lung cancer because the lung cancer of asbestos occurs outside of the scar area (which is located in the lung parenchyma). (CX 17, p.15).

The physician stated that there was a synergism, or multiplier effect, where a smoker was exposed to asbestos. Dr. DeGraff testified that in this case

> The fibrosis was also associated with presence of ferruginous bodies, and in the presence of ferruginous bodies and with fibrosis, you have to consider that this is caused by the asbestos. (p. 23).

Dr. DeGraff acknowledged that in his report (CX 1) he reviewed records but not X-rays or CT scans, or tissue samples. Dr. DeGraff gave greater weight to the opinion of Dr. Mark as that physician recut slides and Dr. Cagle did not request those slides. Dr. DeGraff stated that there was no indication that Dr. Cagle had recut slides. (pp. 27 & 28).

Dr. DeGraff stated that in the presence of a history of heavy asbestos exposure and in the presence of ferruginous bodies, the presumption is that the pulmonary fibrosis is caused by asbestos exposure. (pp. 58 & 59).

Without ferruginous bodies ...

> In the presence of just the simple history of heavy exposure to asbestos and the presence of pulmonary fibrosis, the presumption is more likely than not – and that's a legal assumption —— the assumption is that that is asbestosis. (P.59).

The physician was asked about

> a report of the Pneumoconiosis Committee of the College of American Pathologists and the national Institute for Occupational Safety and Health. And it's found at the Archives of Pathological Laboratory Medicine, Volume 106, October 8, 1982. "The demonstration of asbestos bodies in the absence of fibrosis is insufficient evidence to justify the diagnosis of asbestosis. Conversely, the definitive diagnosis of asbestosis cannot be made in cases that show characteristic fibrosis in the absence of asbestos bodies even in a patient with a history of exposure."

Dr. DeGraff responded

> A. The issue is a definitive diagnosis as opposed to more probable than not, and I think we're dealing with more probable than not here. But also, here we have pulmonary fibrosis and we have asbestos bodies and we have a history of exposure. So you have three things which would lead you to the diagnosis of asbestosis. And in any case, my opinion is that asbestosis is a separate issue. The patient has lung cancer. He has a heavy asbestos exposure. The asbestosis only indicates the degree that he had heavy asbestos exposure. The lung cancer develops independent of the pulmonary fibrosis. It is not a scar cancer. It develops within the airways. (pp. 60 & 61).

Reference was made to the X-ray B-reader's diagnosis of asbestos related disease. Dr. DeGraff indicated that pulmonary fibrosis was present whether or not the term "asbestosis" was used. The physician stated that pulmonary fibrosis is a marker for high levels of asbestos exposure. (pp. 64 & 65).

Dr. Jones was deposed in July 2002 and testified that there must be a diagnosis of asbestosis before lung cancer could be attributed to asbestos exposure. (DX 8, p. 11). The physician stated that

> Asbestosis is a diffuse scarring deep within the substance of the lung. It's a linear or non-nodular type of scarring and it's caused by inhalation of moderate to heavy concentrations of asbestos over a long period of time, usually decades.
>
> ... There are two principal ways to diagnose asbestosis. One is the clinical or radiologic-based method where lung tissue is not available. And the other is a histopathologic method when there is available suitable lung tissue for the determination. (p. 16).
>
> ... Interstitial fibrosis just means scarring within the meaty substance of the lung. And that is one of the two elements of histopathologic diagnosis, the other being multiple asbestos bodies in association with that interstitial fibrosis. (pp. 23 & 24).

Dr. Jones reported that he relied on the 1982 CAP/NIOSH standards. The physician also stated that an X-ray diagnosis could be based on findings of interstitial lung disease.

Dr. Jones reviewed records as well as X-rays and CT scans. The physician stated that

> Mr. Sauls did not have asbestosis based on everything I've reviewed, and therefore, his lung cancer is not attributable to such asbestos exposures as he may have had. (p. 32).

Sauls did not have diffuse lung disease. (p. 33). There was an absence of radiologic evidence of asbestosis. (p. 35). In addition, the pathologists did not assert such a diagnosis. (P. 36).

Dr. Jones was asked about Dr. Pohl's report. Dr. Jones stated that

> You notice that he said only that it was consistent with pulmonary asbestosis at that point. But, of course, the same kind of fibrosis is consistent with dozens and dozens of other causes. To make a diagnosis of asbestosis from tissue slides, you have to have what he described plus you have to have multiple asbestos bodies. (p. 46).

In reference to Dr. Mark's report, Dr. Jones stated he believed that Dr. Mark did not make a diagnosis of asbestosis as the tissue samples would not support such a diagnosis. (pp. 47 & 48).

> Dr. Pohl's report is not really credible. He refers to Mr. Sauls' asbestosis as if he had diagnosed it, when without asbestos bodies he's not entitled to assert a diagnosis.
>
> But I thought that Dr. Mark probably would; but when I received his report and saw that he did not, this is less of a dispute between him and Dr. Cagle than I had imagined it would be. (p. 49).

Dr. Jones did not concur in the B-reader's findings as a CT scan did not show diffuse lung disease or pleural plaques. In addition, Dr. Jones felt that Dr. DeGraff assumed that a diagnosis of asbestosis was established after reviewing the reports of Drs. Pohl and Mark. (p. 53).

Dr. Cagle cut lung tissue and did not find asbestosis. Dr. Jones concurred with Dr. Cagle in the opinion that asbestosis must be present in order to relate lung cancer to asbestos exposure. (pp. 54 & 55).

The physician stated that

> The gold standard is the histopathological diagnosis and the reason is that, as I've testified, what you see on the x-ray that you infer is asbestosis in the presence of other information is simply interstitial abnormality. What the pathologist sees when he looks at tissue sections is actual scarring or fibrosis, and therefore, the pathologic examination is more specific for the fibrosis. (p. 57).
>
> Fibers are cleared from the lung as a result of several processes. There's no question. But for histopathologic diagnosis, you still have to have asbestos bodies in association with fibrosis. And if they're not found, then it's not asbestosis according to this document. [CAP/NIOSH] (p. 83).

Dr. Jones noted that while Drs. Pohl and Mark stated that Sauls had interstitial fibrosis, Dr. Cagle found otherwise. Dr. Jones felt that Dr. Pohl did not follow the CAP/NIOSH standards as to a diagnosis. While Dr. Mark found multiple asbestos

bodies, he did not say that he found these in association with fibrosis, and Dr. Mark did not make a diagnosis of asbestosis. (DX 8, p. 95).

Dr. Cagle was deposed in August 2002 and testified that he specialized in lung pathology. The physician reviewed slides as well as sections cut from tissue blocks. Records indicated that Sauls had a heavy history of cigarette smoking.

Regarding asbestosis, the physician stated that

> The diagnosis involves looking at an adequate sample of lung tissue in a tissue section under the light microscope and observing the characteristic pattern of fibrosis that I referred to, combined with finding the asbestos bodies. As a bare minimum, we expect in a case of asbestosis to find mature fibrosis in the walls of the air sacks that make up the wall of respiratory bronchioles and at least two asbestos bodies. Generally we will find more, and the more fibrosis we will find more asbestos bodies. (DX 9, p. 18).

An iron stain on a tissue sample would make an asbestosis body which is an iron-coated asbestos fiber stand out. Dr. Cagle did not find any asbestos bodies. (p. 22). The examination did not show the characteristic pattern of fibrosis one would expect with asbestosis. (p. 28).

Dr. Cagle reviewed photographs taken by Dr. Mark and made part of Dr. Pohl's deposition. Dr. Cagle agreed that photograph 1D could reflect an asbestos body. The physician did not mention asbestos bodies on reviewing photographs 1E through 1I. Dr. Cagle stated that

> It is not required that if one has a cancer that's caused by asbestos exposure that the asbestos bodies be in the tumor. That's not required. So I would, as I did in Mr. Sauls' case, in looking for asbestos bodies, look not only in the areas with the tumor, but I would especially look in other areas, because as the tumor grows, that's new growth, that may push away something that had been there like an asbestos body. (p. 35).

Dr. Cagle stated that he did not see slides form Dr. Mark or Dr. Pohl. The one slide in picture 1A - 1D did show one asbestos body. The physician agreed, in essence, with Dr. Churg's definition of asbestosis. Dr. Cagle would disagree with an

opinion that asbestos could be diagnosed in the absence of asbestos bodies in the tissue (presumed reference to the Helsinki Consensus Standards).

The physician reported that an asbestos body is an asbestos fiber that has been coated with material such as iron. In addition,

> The presence of the asbestos bodies in terms of the disease asbestosis do not mean anything only by themselves. One must also then find the characteristic fibrosis in a pattern that is diagnostic of asbestosis within the presence of sufficient asbestos bodies to account for that characteristic pattern of fibrosis. (p. 52).

Dr. Cagle acknowledged that some inhaled asbestos fibers underwent dissolution and fragmentation and were removed from the lungs. They physician did agree with the CAP/NIOSH report where it stated that because asbestos bodies are unevenly distributed in tissue, an adequate number of samples should be examined. (p. 61).

An attorney pointed to an article by Dr. Churg where that physician indicated that a single body in combination with the correct pattern of diffuse interstitial fibrosis was diagnostic of asbestosis. Dr. Cagle stated that an asbestos body might be randomly encountered, and that the best solution was to examine multiple sections. Photograph 1D was consistent with an asbestos body but the other pictures from Dr. Mark/Dr. Pohl were not. (DX 9).

Dr. Cagle completed a report in October 2002. The physician stated, in part

> An asbestos body count of 150 asbestos bodies per gram weight wet lung tissue (AB/gmwwt) was obtained after digestion of a sample of Mr. Sauls' lung tissue. This concentration of asbestos bodies is above our background level (100 AB/gm-wwt) and, therefore, does indicate that Mr. Sauls had an exposure to asbestos above background consistent with his work history. However, this asbestos concentration is too low to cause asbestosis (requires 1000 AB/gm-wwt). This asbestos concentration is too low to cause a risk of asbestos-related lung cancer (requires 1000 AB/gm-wwt). Indeed, this asbestos concentration is too low to be likely to be seen in routine slides of tissue sections

which is consistent with the absence of asbestos bodies in H&E or iron stained tissue sections from Mr. Sauls' lungs (generally requires about 250 AB/gm-wwt).

Therefore, the concentration of asbestos bodies in Mr. Sauls' lungs on digestion study is consistent with his work history and with the findings on H&E and iron stained sections of his lung tissue as stated in report BYC2002-032. The concentration of asbestos bodies in Mr. Sauls' lungs confirms that Mr. Sauls was not at risk for an asbestos-related lung cancer consistent with the findings on H&E and iron stained tissue sections from Mr. Sauls' lungs as stated in report BYC2002-032.

In Summary:

(1) Within reasonable medical probability, at a concentration of 150 AB/gm-wwt, the fibrous tissue seen in Mr. Sauls' lungs was not caused, contributed to or aggravated by exposure to asbestos.

(2) Within reasonable medical probability, at a concentration of 150 AB/gmwwt, Mr. Sauls' lung cancer was not caused, contributed to or aggravated by exposure to asbestos. (DX 10).

The Report of the Pneumoconiosis Committee of the College of American Pathologists and National Institute for Occupational Safety and Health, 106 Arch Pathol Lab Med, No. 11, October 8, 1982, was submitted as JX 1. The committee chairman was Dr. Craighead, and Dr. Churg was on the panel. The paper is commonly known as the CAP/NIOSH report.

## Discussion

The undersigned does not claim to have medical expertise, and if I were knowledgeable in this field, the rules would prohibit me from using such information. The undersigned did preside in a similar case in Newport News Shipbuilding & Dry Dock Co. v. Parks, which was ultimately decided by the U. S. Court of Appeals for the Fourth Circuit in an unpublished opinion in 1999 (No. 98-1881).

There is a battle among experts as to the definition of asbestosis in this case. Dr. Mark, a pathologist, has submitted

two reports, CX 14 and CX 3. In CX 14, this physician reported reviewing 36 slides and then cutting 6 slides from a tissue block. He found asbestos bodies in four of six slides, with at least one body per square centimeter. Asbestosis was not specifically diagnosed but Dr. Mark related the lung cancer, in part, to the ingestion of asbestos fibers.

In the later report, Dr. Mark spoke of cancer of the larynx. The physician stated that

> Smoking and asbestos each can cause or contribute to cause carcinoma of the larynx inc1uding squamous cell carcinoma.
>
> I conclude that the patient has developed two squamous cell carcinomas, first one in the lung and then one in the larynx. I conclude that the cigarettes which he reportedly smoked and the asbestos which he inhaled caused both squamous cell carcinomas. (CX 3).

Dr. Pohl, a pathologist, reviewed slides originating at the hospital. On some slides,

> Lung tissue sampled distant from the tumor shows the presence of an advanced well-established interstitial fibrosis consistent with pulmonary asbestosis. The histologic appearance of the tumor is diagnostic for a poorly differentiated adenosquamous carcinoma of bronchogenic origin.
>
> ... Mr. Sauls' asbestos exposure was prolonged and significant, fulfilling the Helsinki Consensus Conference criteria for significant occupational exposure to asbestos.

Dr. DeGraff, a pulmonologist, noted the X-ray reading and the reports from Drs. Mark and Pohl. The physician related that Dr. Pohl had reported that one slide case revealed well, advanced interstitial fibrosis consistent with asbestosis. Dr. Mark had noted fibrosis and asbestos bodies in some of the sections that he had recut. Dr. DeGraff concluded that asbestosis had been documented radiologically and pathologically.

Dr. DeGraff later stated that Sauls had a history of exposure, that there was pulmonary fibrosis, and that asbestos bodies were found. Dr. DeGraff noted that Dr. Cagle did not review the slides cut by Dr. Mark.

In 2002, Dr. Pohl indicated that based on a history of exposure asbestosis bodies need not have been found as the fibers disappear with time. The physician stated that he favored the Helsinki criteria (emphasis on exposure) over the CAP/NIOSH requirement of finding asbestos bodies.

Dr. Pohl listed numerous slides that he reviewed, and Dr. Cagle's list indicates that he looked at all of those. Apparently, Dr. Mark prepared and reviewed other slides and Dr. Cagle performed similarly. Neither physician saw the additional slides prepared by the other. Dr. Mark reports reviewing 35 slides, Dr. Pohl mentions 31, and Dr. Cagle states 34 plus the additional cuts.

Drs. Pohl and Cagle reviewed Case S-98-10234. Dr. Pohl reported well established interstitial fibrosis consistent with pulmonary fibrosis. Dr. Cagle described

> A few microscopic foci of peribronchiolar fibrosis consistent with changes from previous tobacco smoking are present. No asbestos bodies are present on H&E or iron stained sections. Dr. Cagle reported that slides he prepared and the other slides were negative as to asbestos bodies.

Dr. Cagle stated that

> In his report of 8-30-01. Dr Pohl makes mention of interstitial fibrosis in the slides with Mr. Sauls' lung tissue from the lobectomy (S98-10234) that he interprets as "consistent with pulmonary asbestosis." Dr. Pohl does not mention finding asbestos bodies and, indeed, no asbestos bodies are present in the H&E stained slides of Mr. Sauls' lung tissue from the lobectomy.

In early 2002, Dr. Jones deferred to Dr. Cagle. (DX 5).

Dr. DeGraff stated that Dr. Mark had described ferruginous bodies and that a diagnosis of asbestosis was not necessary to relate asbestos exposure to lung cancer.

Dr. Pohl later stated that exposure was more important than findings of asbestos bodies. In mid-2002, Dr. DeGraff supported Dr. Mark's report as that physician had recut slides, and Dr. DeGraff was unaware of whether or not Dr. Cagle had recut slides.

Dr. Jones stated that while Dr. Pohl had reported that fibrosis was consistent with asbestosis, such fibrosis was indicative of many causes.

Dr. Jones stated that Drs. Mark and Pohl had reported asbestosis although there were no findings of multiple asbestos bodies.

Dr. Cagle reported in mid-2002 that he had cut additional slides, although Dr. Pohl understood otherwise. This physician found one asbestos body in the pictures taken by Dr. Pohl. In late 2002, Dr. Cagle reported that a digestion study had revealed a relatively low count of asbestos bodies which was considered below the standard for a diagnosis of asbestosis.

The physicians in this case have reported the number of slides that they reviewed. It is fairly certain that Dr. Cagle did not review the slides cut by Dr. Mark. However, it is certain that Dr. Mark did not review those cut by Dr. Cagle.

Dr. Mark makes a general statement as to the findings of asbestos bodies. He does not make specific designations as to the findings. Dr. Pohl seems to accept statements from Dr. Mark at face value and essentially reports that asbestosis can be diagnosed on a history of exposure to asbestos, alone.

Dr. Cagle cut 5 slides and reviewed some 35 others, and did not make a diagnosis of asbestosis, based on the CAP/NIOSH criteria of finding two asbestos bodies in a size specified sample.

Dr. Cagle is quite specific in his findings and in his conclusions. The CAP/NIOSH criteria seems sounder than that of the Helsinki Committee. The evidence does not reflect that deference must be paid to the Helsinki report. I give greater weight to the opinion of Dr. Cagle than to those of other physicians.

It is concluded that asbestosis has not been proven in this case. Asbestos exposure has not resulted in lung or in larynx cancer.

As Mr. Saul’s claim for benefits is denied there is no reason to address other issues such as responsible employer/carrier and entitlement to Section 8(f) relief.

## ORDER

Mr. Saul's application for benefits based on asbestos exposure is **DENIED**.

A

Richard K. Malamphy
Administrative Law Judge

RKM/ccb
Newport News, Virginia